VII. SENSORY SYSTEM

SOMATIC SENSATION

TRANSDUCTION: The process by which a physical stimulus is converted into a neural signal and sent to the CNS.

- **Trigger Zone:** The threshold of stimulus, in the sensory receptor, at which an action potential is generated. Some sensory receptors are more sensitive than others.
- **MODALITY SPECIFICITY**: Any particular sensory unit is most sensitive to only one modality. • There are four broad classes of somatic stimuli:
- The modality to which a receptor it is sensitive is called the **adequate stimulus** for the receptor.
- The specific modality is the one that triggers the receptor at the *lowest threshold potential*. Other modalities may also trigger the receptor, but at much high potentials.
- Paradoxical Cold is an exception to the Modality Specificity rule.
- Sometimes heat may be perceived as cold, because it triggers cold fibers rather than warm fibers.
- Normally warm fibers are triggered by an increase in temperature, and cold fibers by a decrease in temperature.

FIBER DIAMETER AND MODALITY SPECIFICITY:

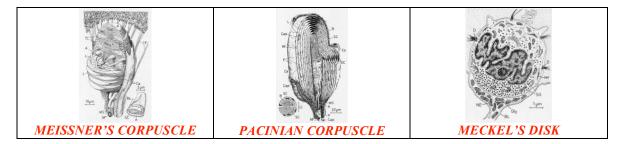
- Class II (A-β) Fibers: Cutaneous Sensation
- Fibers terminate in specialized nerve endings such as Merckel's Disks and Pacinian Corpuscles.
- ASPHYXIA: These fibers are most sensitive to asphyxia and to physical insult, because they are the largest of the sensory fibers.
- Anesthesia: These fibers are the *last* to be blocked by anesthesia—they are the largest fibers.
- Class III (A-δ) Fibers: *Fast Pain*, crude touch, temperature sensation.
 - o Fibers terminate in free nerve endings.
- FAST PAIN: Pin-prick pain; it is the first pain you will feel when pricking your finger.
- Class IV (C) Fibers: Slow Pain, crude touch, temperature sensation.
 - o Fibers terminate in free nerve endings.
 - SLOW PAIN: Throbbing pain, which evokes the troublesome affective experience of pain.
 - ANESTHESIA: Slow pain fibers are the most sensitive to local anesthesia. Anesthesia blocks smalldiameter fibers before large-diameter.
 - Asphyxia: These fibers are the *last* to be blocked by asphyxia, as they are the smallest fibers.

CLASS	DIAMETER VELOCITY	ELECTRICAL STIMULATION	SENSATION
Π (A-β)	Relatively large diameter Fast conduction velocity	Lowest Threshold (i.e. first to be stimulated)	Cutaneous Sensation
III (A-δ)	Small diameter Slow conduction velocity	Medium Threshold	Fast Pain Crude Touch Some temperature
IV (C)	Smallest diameter Slowest conduction velocity	Highest Threshold (i.e. last to be stimulated)	Slow Pain Crude Touch Some temperature

FREE NERVE ENDINGS: Pain and temperature both end in free nerve-endings in the skin.

SPECIALIZED NERVE ENDINGS: They mediate tactile sensation: flutter, vibrations, and pressure.

- **MEISSNER'S CORPUSCLES:** They mediate the sensation of **flutter**—localized, slow vibrations.
 - Rapidly-Adapting, Phasic response: The receptor shows Adaptation in that it stops firing after the same stimulus has been present for a while. It "blocks" out the stimulus once the stimulus becomes old news.
 - o Anatomical Distribution: Found in Glabrous (non-hairy) skin, as in palm of hands.

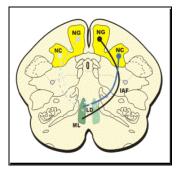


- PACINIAN CORPUSCLES: They mediate the sensation of vibration.
- **Rapidly-Adapting, Phasic** response: The receptor stops firing after the stimulus has been present for a while.
- STRUCTURE: It is like an **onion**.
- MECKEL'S DISKS: Mediate sensitivity to pressure.
- Slowly-adapting, Tonic response. The nerve continues to discharge as long as the stimulus remains. So, you continue to feel pressure as long as the pressure is still there.

ADAPTATION: "A decrease in neural response to sustained stimulation." Meissner's and Pacinian corpuscles both show adaptation.

- PHASIC RESPONSE refers to the fact that the receptor will fire only when there is a *change in* stimulation.
- TONIC RESPONSE: Refers to continual firing of the receptor when a continual stimulus is present. No change in stimulus is necessary to maintain firing.

DORSAL-COLUMN MEDIAL-LEMNISCAL PATHWAY: *Proprioception and Discriminative Touch* run parallel with each other but actually have separately named paths.



- PATHWAYS: For spinal components (not trigeminal). *These fibers enter through the medial branches* of the Dorsal Horn.
 - o DISCRIMINATIVE TOUCH: Discriminative Touch fibers are Group II (A-β) fibers.
 - So, Area 3B receives 3rd-order neurons originating from specialized receptors (Meckel's Disks), via Group II fibers
 - PROPRIOCEPTION: Conscious Proprioception receptors are 1A-SPINDLE FIBERS
 So, Area 3A receives IIA Spindle Afferents for proprioception signal.
- SOMATOTOPIC ORGANIZATION
- Spinal Cord: Sacral is most medial and Cervical is most lateral. As you move up the cord, sacral segments enter the cord first, and higher up segments enter right "on top of," i.e. lateral to, the sacral segments.
- o THALAMUS: Somatotopic Organization is essential reversed.
- SOMATOSENSORY CORTEX: There are *four relatively complete maps* of the body: 3a, 3b, 1, and 2.
- DISTORTED REPRESENTATION: Finger tips, lips, and tongue get a disproportionate amount of cortex, because they are the most sensitive sensory organs.
- DESCENDING SENSORY Connection: Descending *Sensory* fibers go from Sensory Cortex -----> Thalamus -----> Dorsal Column Nuclei. They may serve a role in adaptation or filtering repetitive stimuli, but function is unsure.
- LESIONS:
- **Tabes Dorsalis:** Secondary to Syphilis; lesion of dorsal columns. Patients show deficits in proprioception and discriminative touch, but not pain and temp.
- Transection of Dorsal Columns: Results in large increase in two-point discrimination.
- Destruction of S1 (Somatosensory Cortex): Expected sensory deficits result.

Labelled Line Theory: There are separate pathways for each modality of sensation, and these all run into the CNS in a parallel fashion.

- **CORTICAL COLUMNS:** Each column contains layers that represent *different modalities*, but they all came from the *same region of the body*.
 - o So, neurons in the same "layer" or lamina of the cortex will exhibit the same modality specificity.

RECEPTIVE FIELDS: The area of skin which, when appropriately stimulated, causes a neuron to discharge.

- The smaller the receptive field, the more sensitive is the sensory ability. Smaller receptive fields mean higher acuity.
- Proprioceptive and Discriminative Touch (DC-ML) fibers have smaller receptive fields than pain and temp (anterolateral) fibers.
- **TWO-POINT THRESHOLD:** The minimum distance, on the skin, at which two pin-points can be distinguished. The smaller the two-point threshold, the higher the *tactile acuity*.
- Fingertips and lips have smallest two-point-thresholds (~2 mm); trunk has a larger threshold (~60 mm)
 Small receptive fields correspond to high innervation density and a disproportionately large amount of somatosensory tissue in the CNS.

PHANTOM LIMB SYNDROME: People who have had a severed limb still retain sensation that the limb is there (proprioception) when other parts of the body are stimulated, such as the face.

• This is due to **plasticity** of neurons in the CNS. The CNS neurons that used to supply the missing limb are near the face, so they can get stimulated when face is stimulated.

HIERARCHICAL PROCESSING: As one ascends through the CNS, more complex types of sensations are processed.

- Area 3a and 3b (aka S1) are the first recipients of sensory information.
- Area 1 and 2 (aka S2 (?)) receive input from Areas 3a and 3b. Thus 1 and 2 are higher up in the processing of somatic sensation.
 - For example, Areas 1 and 2 can discriminate selectivity of movement of a finger across the skin, whereas area 3 cannot.
- AREA 2: It is unique in that it receives a *convergence of multiple modalities*. Both Proprioceptive and Tactile input can arrive at the same fibers.
 - This convergence of function allows for Area 2 to perform **Stereognosis** (identifying objects by touch). *Area 2 lesion results in severe deficit in this specific ability.*

NEGLECT SYNDROME: Lesion of posterior Tempero-parietal area. **Extinction** occurs in the Neglect Syndrome. Extinction is failure to recognize a specific stimulus (either visual, somatic, and/or auditory) on one side of the body, contralateral to the lesion.

PAIN

- Terms:
 - Nociceptive Stimuli: Stimuli that produce pain.
- Analgesia: A condition in which nociceptive stimuli are not perceived as painful.
- Two components of Pain: The two components of pain are separable by drugs.
 - The sensation of pain itself.
- The affective component of pain in which it is perceived to be painful or unpleasant.
- **Morphine** separates these two components such that patients still feel the pain but they do not find it to be unpleasant or "painful."
- **PAIN-PRODUCING STIMULI (PPS)**: Chemicals that are involved in transduction of *slow-pain* fibers.
 - **POTASSIUM**: High extracellular K⁺ is indicative of tissue damage and is therefore painful.
 - **BRADYKININS:** Tissue injury -----> proteolytic enzymes into the extracellular fluid -----> react with gamma globulins to create Bradykinins.
 - Aspirin will block this prostaglandin synthesis, wouldn't you know??
 - HISTAMINE: Substance-P, released by C-fibers, causes Mast Cells to release Histamine.
- Neurotransmitters used in Anterolateral System:
 - o GROUP III (A-delta) NEUROTRANSMITTER: Glutamate
 - GROUP IV (C-FIBER) NEUROTRANSMITTERS: C-Fibers have two neurotransmitters which cause vasodilation when released on vessels.

- ANTEROLATERAL SYSTEM PATH: Group-III (Fast Pain) and IV (slow-Pain) fibers enter the spinal cord through the *lateral portion* of the Dorsal Root over the **Tract of Lissauer** -----> Ascend one or two segments -----> Synapse in **Substantia Gelatinosa** -----> CROSS -----> Ascend in Anterolateral tract.
- NEOSPINOTHALAMIC PATHWAY: Fast pain (III) and temperature sensation.
 - *Fast Pain only* goes through the Marginal Zone.
 - The fibers split into layers in the Tract of Lissauer.
- PALEOSPINOTHALAMIC PATHWAY: Receives Slow-Pain (IV) fibers, plus some Fast-Pain.
 Lesion of the Intralaminar Nuclei will relieve chronic pain.
- Anterolateral Cordotomy: Sectioning the anterolateral cord on the *contralateral side* in order to relieve intractable pain.
- Targets of Anterolateral Pathway:
 - Spinoreticular Pathway: Also for modulation of pain (see below)
 - Spinotectal Pathway: Also involved in pain control; orientates our response to painful stimuli.
 - o Spinothalamic Pathway: The primary pathway for pain transmission to Thalamus.

TRIPLE RESPONSE OF LEWIS (AXON REFLEX):

- Wheal: Localized raised area resulting from vasodilation from local irritants.
- Flare: Reddened area surrounding the wheal.
- It is an *axon-axon reflex* that does not go through the CNS. Local Nociceptive fibers are stimulated, and they send messages to neighboring fibers to cause a "flare" of vasodilation around the original wheal.
- **Capsaicin** is a peppery substance that causes the wheal-and-flare response locally. Applied continually, it will *desensitize the C-Fibers* to local allergens and can thus be used as a topical analgesic.
- **HYPERALGESIA:** Enhanced sensitivity to pain occurs in the region following the Wheal and Flare response.

THALAMIC (CENTRAL PAIN) SYNDROME: Spontaneous pain, and exaggerated responses to pain stimuli, resulting from a *vascular lesion in the Thalamus*.

- Triad of related symptoms:
 - o Spontaneous Pain
 - o Non-Injurious stimuli (light touch, movement) are perceived as painful.
 - o Hyperalgesia: aggravated pain response.
- Originally, it was thought that only the Thalamus produced these symptoms, but it is now known that a lesion anywhere along the pain pathway (such as anterolateral cordotomy) can produce the symptoms.

MECHANISMS OF ANALGESIA

- GATE CONTROL THEORY: Transmission of pain information can be modified by descending CNS large-fibers. *Endogenous activity in large-fiber pathways can block pain*.
- After you bump your head, rubbing it can help it. When you rub your head you are stimulating large fiber pathways.
- **Spinotectal and Spinoreticular Pathways**: These are ascending pathways that in turn lead to *inhibition of pain transmission in the dorsal horn*. These pathways are an endogenous way of modulating pain.
- *Stimulus0Produced Analgesia* can occur from electrical stimulation of the **periaqueductal gray**. Again, this analgesia has its effect by inhibiting pain transmission in Dorsal Horn.
- OPIOIDS:
 - o Inject very small amount of Morphine into one of two CNS regions to cause profound Analgesia:
- Opioid Receptors: **Enkephalins** and **Endorphins** are the endogenous ligands for these receptors. Opiates also bind to them but with higher potency.
- **Naloxone** is an antagonist to this receptor.
- STRESS-INDUCED ANALGESIA: Extreme stress (Epinephrine) can induce analgesia so that a person can perform actions that would normally be painful. The action is not perceived as painful until after the stressful event is over.

RADICULAR PAIN: Pain localized to the dermatome of a dorsal root.

- Injury to a single Dorsal Root will not usually produce Anesthesia, because there is overlap between dermatomes.
- Paresthesia (tingling, etc.) is common however, and Radicular Pain often occurs with Paresthesia.

REFERRED PAIN: Visceral injury will send afferent pain information on the same nerves that also serve a cutaneous region. Because the brain is more used to getting sensory input from the cutaneous region of the nerve, the CNS will interpret the pain as originating from the cutaneous region.

VISION

CONVEX LENS: Converging rays. They shorten the focal length and can be used to correct for farsightedness.

• Positive Focal Length—focal point is in front of the lens.

CONCAVE LENS: Diverging rays. They lengthen the focal lengthy and can be used to correct for nearsightedness.

• *Negative focal length*—focal point is behind the lens.

LENS POWER: It indicates how much the lens can converge or diverge the light rays. *The stronger the power, the shorter the focal length*. A short focal length means the light is being bent a lot.

EMMETROPIC EYE: Normal vision, in which the light rays form an image on the retina.

- The natural eye has *two convex lenses*, which serve to focus the light on the retina.
 - Cornea
 - o Crystalline Lens
- The image on the retina is a "real" image—upside down and inverted.
- ACCOMMODATION: A relaxed lens is relatively flattened and lets you focus at a distance. Accommodation increases the curvature of the lens to focus for near vision.
- Relaxed lens lets you focus at 63 mm from the surface of eye.
- Cycloplegics are topical drugs you can put on the eye to inhibit the accommodation reflex and cause the lens to relax.
- **PRESBYOPIA**: The loss of accommodation ability occurs with age. Children have up to 14 diopters of power to accommodate, while old adults may have only 1 or 2 diopters, or none.

MYOPIA, **NEARSIGHTEDNESS**: You can see things up close just fine but have difficulty seeing into the distance.

- The image forms in front of (anterior to) the retina.
- Corrective Lens: **Diverging (Concave) Lens**, to lengthen the focal length and move the image back a little so it forms on the retina.
- Other corrective procedures:
 - **Radio Keratotomy (RK)**: Use fine diamond scalpel is used to make radial incisions in cornea, in order to flatten the cornea itself.
 - **Photorefractive Radio Keratotomy (PRK):** UV laser is used to blow off pieces of the cornea, a bit at a time.

HYPEROPIA, FARSIGHTEDNESS: You can see things far away just fine but have difficulty seeing things up close.

- The image forms behind (*posterior to*) the retina.
- Corrective Lens: Converging (Convex) Lens, shorten the focal length and move the image forward a little so it forms on the retina.
- Other corrective procedures:
 - o Laser Keratothermoplasty: Surgery that increases the curvature of lens.

ASTIGMATISM: A problem in which the *cornea has two different radii of curvature (one horizontal and one vertical) and two different focal points*. Essentially, the cornea is deformed such that the horizontal and vertical focal lengths don't match up with each other.

- Torus is donut-shaped thingie that has two radii of curvature: R₁ is the radius of the donut itself, and R₂ is the cross-sectional radius of the donut.
- Corrective Lens: **CYLINDRICAL LENS**. *The cylindrical lens is curved only in the horizontal plane*—*not in the vertical plane*.
 - There is no power in the vertical direction.
 - Regular lenses can't be used because the lens would correct for the problem in one direction, only to cause a distortion in the other direction.
 - After correction, the patient may still be highly myopic. This can be further corrected with a normal spherical lens.

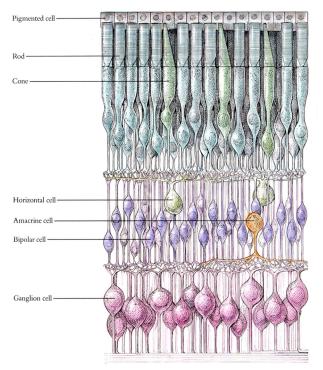
VISUAL ACUITY: The minimal angular resolution of the eye.

- **beta** = *the minimum angle of resolution of two dots*. The minimum subtended angle at which a patient can perceive two dots as distinct.
 - \circ Minutes of arc are used to measure this angle, where 60 minutes = 1 degree.

- Limit to Visual Acuity: The ultimate (theoretical) limit to visual acuity are the photo-receptors of the eye.
 - In the fovea (finest visual acuity), photoreceptors are separated by about **3 microns**, which corresponds to a theoretical minimum subtended angle of **0.6 minutes**. This theoretical minimum is never attained due to some **diffraction** of light in the eye.
- Factors that affect Acuity:
- Pupil size. Small pupils result in better visual acuity.
- SNELLEN EYE CHART: Made of strokes and gaps, which in standard form should be separated by 5 units from each other.
- 20 / 20 (x / y) vision:
- NUMERATOR: The distance at which the patient stands from the chart. It is basically always 20!
- DENOMINATOR: The *distance at which the letter would subtend 1 minute of arc for this patient*. This is equivalent to the distance at which an emmetropic eve could view the letter.
- Due to the above, the relationship between minutes of arc and the Snellen Eye Chart is that the distance = (20)(minutes of arc).

OPTICAL AXIS: Straight through the lens. It is slightly medial to the Visual Axis, which is where the fovea is located.

THE RETINA: From the back of the eye (outer limit of eye) to the inner most layer...



- RETINAL EPITHELIUM: Not officially part of the retina.
 - Choroid—Not officially part of the retina. It is the blood supply to the retina.
- **Retinal Pigmented Epithelium**—Not officially part of the retina, support and recycling of rhodopsin disk-membranes.
- PHOTORECEPTOR LAYER
 - **OUTER SEGMENT**: Photoreceptor Cells, containing the photosensitive elements.
- **INNER SEGMENT LAYER:** Continuation of Photoreceptor Cells, containing the mitochondria and other support organelles.
- OUTER LIMITING MEMBRANE really isn't a membrane, but consists of tight junctions between the cytoplasmic extensions of Muller Cells. It contains MULLER CELLS, which are Retinal Glial cells.
 - o Muller cells have their end-feet on both the outer and inner limiting membranes.
 - \circ They regulate the extracellular K⁺ concentration in the environment.
- They contain *Glutamine Synthetase*, necessary for metabolism of Glutamate, which is the main excitatory neurotransmitter in the visual system.
- OUTER NUCLEAR LAYER: Contains the *cell-bodies* of the photoreceptors.
- **OUTER PLEXIFORM LAYER**: Contains *synaptic connections* between the photoreceptor-cells and the integrating neurons (amacrine and horizontal cells).

- **INNER NUCLEAR LAYER**: Contains the *cell-bodies of the integrating cells*. There are three integrating cell types:
- Amacrine Cells
- o Horizontal Cells: Process lateral information.
- Bipolar Cells: *This is the basic second-order neuron*. The "Standard Synapse" is photoreceptor cell ----- --> bipolar cell -----> ganglion cell.
- o Muller Cells: This cell extends almost the entire length of the retina.
- **INNER PLEXIFORM LAYER**: Contains synaptic connections between the integrating cells and the Ganglion Cells.
- GANGLION CELL LAYER: Contains the cell-bodies of the Ganglion Cells. They're afferent fibers make up the optic tract.
- NERVE FIBER LAYER: Unmyelinated axons of Ganglion cells.
- INTERNAL LIMITING MEMBRANE: Separates the neural retina from the Vitreous Body.

OPTIC DISK: Region of retina where the optic nerve and blood vessels enter. The optic disk is a blindspot, but it is in a different part of the visual field for each eye so normally (with both eyes open) the blindspot is not evident.

• You can see the Optic Disc through the Ophthalmoscope, and this is where you go to test for vascular problems or CNS problems, such as **Papilledema**, which indicates CSF buildup.

FOVEA: Contains no vessels, no inner-nuclear layer, no ganglion cell layer, and *no rods*. Just a high concentration of **cone-photoreceptors**.

PRIMARY PHOTO TRANSDUCTION:

- THE ROD CELL: Specialized for low-acuity vision in the dark.
- Rod cells are sensitive to blues and purples in the spectrum.
- THE CONE CELL: Specialized for high-acuity vision in the light.
- \circ Red Cone Cell
- Green Cone Cell
- \circ Blue Cone Cell
- **OPSINS:** Rod cells contain Rhodopsin and Cone-cells contain Cone-Opsins.
- Photon of Light CHANGES THE CONFIGURATION of RETINAL from 11-cis-Retinal -----> alltrans-Retinal. This is the chemical basis for photo transduction.
- The *trans*-Retinal then escapes the cone cells and goes back to the epithelium where it will get recycled.
- IN THE DARK: The rod-cell is depolarized.
- High levels of cGMP are in the rod-cells.
- \circ Na⁺-Ca⁺²-Channels are open. The channel accommodates both Na⁺ and Ca⁺², although Na⁺ is the major ion to come in.
- \circ The cell is depolarized as Na⁺ continually comes in, and is pumped back out via Na⁺/K⁺-ATPase located in the inner segment.
- The cell is releasing excitatory neurotransmitter (glutamate, aspartate) onto the secondary neurons.
- IN THE LIGHT: The rod-cell becomes hyperpolarized.
- o cGMP gets cleaved to GMP by phosphodiesterase.
- \circ The Na⁺-Channels close.
- Na/K-ATPase quickly restores the cell to what we normally think of as resting potential—cell is hyperpolarized.
- The cell stops releasing neurotransmitter.

SECONDARY CELLS: They receive signals from the photoreceptors.

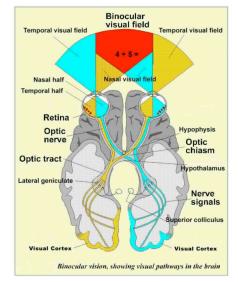
- Bipolar Cells:
 - ON BIPOLAR CELLS: The CENTER of the cell depolarizes in the light.
 - OFF BIPOLAR CELLS: The CENTER of the cell hyperpolarizes in the light.
- CENTER-SURROUND INHIBITION: The region immediately surrounding the center of the cell provides contrast, which makes it easier for the brain to interpret the visual stimuli.
 - o When the center is stimulated, the SURROUND is inhibited by horizontal cells
 - When the SURROUND is stimulated, this indicates that light is nearby but not directly on the center. Thus the center is *inhibited* by horizontal cells.
 - HORIZONTAL CELLS are inhibitory interneurons that provide lateral inhibition to surrounding regions: Photoreceptor -----> Excitatory to Horizontal Cell -----> Inhibitory to the *neighboring* photoreceptor and bipolar regions (i.e. the "surround" area of visual field).

• GANGLION CELLS: Each Ganglion Cell has a **Receptive Field Center**, which corresponds, to specific Bipolar Cells, which in turn corresponds to specific photoreceptor cells.

 \circ ON GANGLION CELLS: They are stimulated by the light, i.e. by On Bipolar Cells.

- **OFF GANGLION CELLS:** They are stimulated by the dark, i.e. by Off Bipolar Cells.
- CENTER-SURROUND ORG:
- X and Y SYSTEM is another way to divide up all the cells in the retina, according to their visual function.
- X-SYSTEM (P) is specialized for high visual acuity and color information.
- Y-SYSTEM (M) is specialized for quick detection of motion, such as turning your head to glance at motion in the periphery.

VISUAL FIELDS



- Visual Field Organization with Retina:
- NASAL HALF: The axons of the nasal (medial) half of the retina cross at the optic chiasm and go into the CNS via the contralateral optic tract.
- **TEMPORAL HALF:** The axons on the temporal (lateral) half of the retina do not cross at the optic chiasm and go into the CNS on the ipsilateral optic tract.
- LEFT VISUAL FIELD: Corresponds to the *nasal half* of your left retina and the *temporal half* of your right retina.
- **RIGHT VISUAL FIELD:** Corresponds to the *nasal half* of your right retina and the *temporal half* of your left retina.
- VISUAL FIELD DEFICITS:
- o Terms:
 - This generally happens with *central lesions*, such as in the Occipital Lobe, or a complete loss of the Lateral Geniculate Nucleus.
 - This happens with Pituitary Tumor.
- TUNNEL VISION: No vision in the periphery, or in the temporal visual fields.
- SUPERIOR QUADRANTANOPIA: MEYER'S LOOP Unilateral damage to the ventral aspect of the Lateral Geniculate Nucleus, which contains a representation of the superior visual field, headed toward the inferior bank of the Calcarine Sulcus.
- HOMONYMOUS HEMIANOPIA WITH MACULAR SPARING: Lesion to the Optic Radiations or Visual Cortex on one side.

LATERAL GENICULATE NUCLEUS:

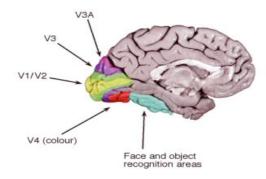
- MAGNOCELLULAR LAYER: Layers 1-2. These layers process Y-Cell information from the retina sudden motion detection.
 - This is the *Where* System, telling you where things are in space. These neurons will ultimately project to the Parietal Lobe to help you orient "where" you are in your visual world.
- PARVOCELLULAR LAYER: Layers 3-6. These layers process X-Cell information from the retina visual acuity and color.
- This is the *What* System. These neurons will ultimately send projections to Temporal Lobe (Wernicke's area, perhaps?) to help you identify what things are.

- Orientation: Different LGN layers process information from different eyes. All of the information from the retina is kept in separate layers in the LGN, so it can be sent to distinct parts of visual cortex.
 - o Layers 1, 4, 6 process the *contralateral* eye.
- Layers 2, 3, 5 process the *ipsilateral* eye.
- Horizontal Orientation: Generally, information in each visual field is represented in contralateral LGN. As you move more laterally in the visual field, its representation moves more laterally in the contralateral LGN.

PRIMARY VISUAL (STRIATE) CORTEX: Area 17.

- Visual Field Organization: Each occipital love processes the contralateral visual field.
- o UPPER QUADRANT of each visual field is on the lower bank of the Calcarine Sulcus.
- LOWER QUADRANT of each visual field is on the upper bank of the Calcarine Sulcus.
- Fovea: The region of highest visual acuity is represented by the most posterior (caudal) part of the Visual Cortex, and it has the largest (disproportionate) representation of neural tissue.
- MEYER'S LOOP: Optic Radiations are the visual track going from LGN to the Visual Cortex. They form Meyer's Loop as they travel around the lateral aspect of the lateral ventricles.
 - o Again, Meyer's Loop lesion leads to visual-field deficit of the contralateral quadrant.
 - Because of its anatomical arrangement, *damage to the temporal lobe can cause superior homonymous quadrantanopia*.
- For similar reasons, damage to the parietal lobe can cause inferior homonymous quadrantanopia.
- CORTICAL COLUMNS: Layers of the Striate Cortex, from Pia to White Matter, are arranged into columns. All visual neurons enter the Visual Cortex through **Layer IV** of the cortex.
- **ORIENTATION COLUMNS:** All neurons in the same column will process will same visualorientation information.
- **EYE-DOMINANCE COLUMNS:** Any particular column will contain exclusively information from the left eye or the right eye.
- **HYPER COLUMNS:** The combination of the Left-Eye Column, Right-Eye Column, plus all possible Orientation-Columns for a single part of the visual field.
- CYTOCHROME BLOBS: Color processing. These blobs are present in each of the hyper columns.

EXTRA STRIATE CORTEX: Visual Association cortices.



- Area V2: The primary extra striate cortex; it receives lots of info from VI, the Striate Cortex. It processes depth information.
- Area V5: Processes information about motion.
- Area V4: Involved in color processing. It receives color information from V2.
- Area IT: Part of the *temporal lobe* involved with recognition of faces.
- Lesion in this area results in **Prosopagnosia**, failure to recognize familiar faces.

Other Retinal Projections: Retinal projection that go somewhere other than the LGN.

- Accessory Optic Nuclei: Sensitive to movement of large visual fields and involved with the nystagmus reflex.
- Suprachiasmatic Nuclei: Part of Hypothalamus; it receives light information in order to influence circadian rhythms.
- Superior Colliculus: Involved with movement of eyes relative to visual stimuli and foveation, focusing, on a moving object.
- There is a sensory map in the superficial layer that has 1:1 correspondence with a motor map in the deep layer. So, when one part of visual field is stimulated, the corresponding motor part directs the eyes to the visual stimulus.
- **Pretectum:** Direct and consensual pupillary light reflexes. It receives bilateral information from the eyes, and projects bilaterally to the Edinger Westphal (CN III) nucleus.

HEARING

Measuring Frequency and Volume:

- SOUND-PRESSURE LEVEL (SPL): A measure of the intensity (volume) of sound in Watts / m². It is related to decibels logarithmically: a tenfold change in SPL corresponds to a linear increase of 20 decibels.
 - \circ If the SPL is one million times (10⁶) stronger, then the number of decibels is 6 x 20 = 120 decibel increase.
- Threshold: The least amount of stimulus energy (SPL) required for the ear to register a sound.
- Threshold is *frequency dependent*. Some frequencies have a lower threshold (are more easily heard) than other frequencies.
- Lowest threshold occurs at 1 3 kHz frequency, which is the optimal pitch at which humans hear.
- **Frequency Threshold Curve** shows the threshold SPL at different frequencies. The thresholds get higher (i.e. more difficult to hear) at high frequencies.
- DYNAMIC RANGE OF HEARING: The range of *volume* that the human ear can detect without incurring damage. The range at 3 kHz is 0 120 dB
- AUDIOGRAM: A frequency-threshold curve, in which the *deficit from normal* is graphed at each frequency range.

EXTERNAL EAR, Pinna:

- It resonates to increase the SPL of the sound between 1 and 3 kHz.
- It plays a role in *sound localization*—its shape aids us in determining where a sound is coming from.

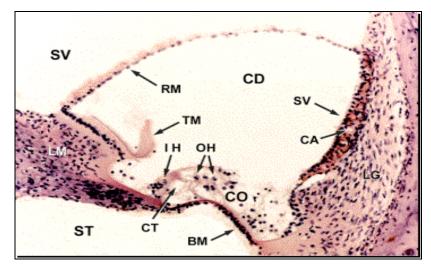
MIDDLE EAR: Air-filled cavity. Malleus -----> Incus -----> Stapes

- Impedance Mismatching: The middle ear bones amplify the sound vibrations from the tympanic membrane to the oval window.
- This amplification of sound gives us a **15 dB advantage**. Removal of middle-ear ossicles does not result in deafness, but rather results in a 15dB hearing loss.
- Tensor Tympani and Stapedius contract reflexively in response to high intensity sound.
- Middle Ear Pathologies:
- Otitis Media: Watch it with children. It will severely impair their language acquisition if they are chronically hard of hearing during those formative years.
- **Otosclerosis:** Conductive Hearing Loss. Stapes no longer vibrates properly against oval window due to abnormal bone growth.

COCHLEA:

- MODIOLUS: The central "shaft" of the Cochlea, around which it screws.
- The modiolus contains the Cochlear Nerve (VIII)
- SPIRAL GANGLION: The starting point of the VIIIth nerve, it is in the Modiolus at the base of the Spiral Lamina.
- SCALA MEDIA: Endolymph fluid similar in composition to intracellular fluid (high in K⁺ and low in Na⁺). The Scala Media contains the sensory hair cells and the Organ of Corti
- o TECTORIAL MEMBRANE is inside the scala media, right on top of the hair cells.
 - The Tectorial Membrane tends to move in an opposite direction as Basilar Membrane. This aids in the shearing force.
 - SOUND TRANSDUCTION: This shearing force transduces the mechanical sound wave into an electrical stimulus.
- STRIA VASCULARIS: Highly vascular epithelium forming one wall of the Scala Media. It secretes endolymph into the Scala Media.
- SCALA TYMPANI: Perilymph fluid. In cross section, it is the section below each Scala Media, below the Basilar Membrane.
- **BASILAR MEMBRANE:** It separates the Scala Media from the Scala Tympani. It forms the base of the Scala Media.
- SCALA VESTIBULI: Perilymph fluid. In cross section, it is the section above each Scala Media, above the Vestibular Membrane.
 - VESTIBULAR (REISSNER'S) MEMBRANE: Very delicate membrane separating the Scala Media from the Scala Vestibuli.
- The Scala Vestibuli is continuous with the **Oval Window.** It therefore conducts sound waves, through perilymph, toward the apex of the Cochlea.
- **HELICOTREMA:** A hole at the *apex* of the cochlea. It connects the Scala Vestibuli to the Scala Tympani

ORGAN OF CORTI: It is located in the Scala Media, a top the Basilar Membrane.



- **INNER HAIR CELLS:** They are closer to the Modiolus. They are very tightly held into place. • STRUCTURE:
 - FNXN: These cells are the **primary sound receptors**. They respond to shearing movements of the Tectorial Membrane.
- o Inner Hair Cells send primary VIIIth Afferents into the CNS, via the Spiral Ganglion.
- OUTER HAIR CELLS: There are more of them, located laterally, away from the tectorial membrane.
- STRUCTURE: They have more room to breathe-there is open space laterally.
- FNXN: These cells can move the *basilar membrane* and can "tune" the frequencies of the basilar membrane.
 - POSITIVE FEEDBACK: The OHC's serve to *amplify* quiet sounds by making the Tectorial Membrane more sensitive to shearing.
 - NEGATIVE FEEDBACK: The OHC's serve to *dampen* loud sounds by making the Tectorial Membrane less sensitive to shearing.
- DISEASE: Outer Hair Cells are subject to disease a lot. Commonly, the OHC's will be missing in a person but the inner hair cells will still be intact. Sensorineural hearing loss, but not total deafness, results.
- **OTOACOUSTIC EMISSION:** Energy emitted by the Outer Hair Cells, which is a real sound and can be detected by the Cochlea. When such emissions are detected, Objective Tinnitus results.
- **Objective Tinnitus:** Ringing in ear when a sound wave is actually present, being generated internally.
- \circ Subjective Tinnitus: Ringing in ear when no sound wave is present.

FREQUENCY SELECTIVITY: Sound is mapped to different parts of the Cochlea according to frequency. Each part of the cochlea is most sensitive to a small range of frequencies, i.e. it has the lowest threshold.

- BASE: Outer part of Cochlea transduces high frequency waves.
 - \circ It is stiff and narrow, helping it to detect high frequencies.
- APEX: Inner part of Cochlea transduces low frequency waves.
- Flaccid and broad, helping it to detect low frequencies.
- Both systems are also energy dependent. Frequency Selectivity still occurs with anoxia, but it isn't as well tuned.
- **Tuning Curve:** If you were to plot the frequency of sounds traveling on any particular VIIIth fiber, you would find that individual VIIIth afferents are also frequency selective, according to which hair cells they innervate.

ENDOLYMPHATIC POTENTIAL: +80mV Potential. The Scala Media is *positive* with respect to the hair cells, and with respect to the perilymphatic compartments.

- DEPOLARIZATION: Inner Hair Cell Stereocilia move toward Kinocilium ----> Open K⁺-Channels ---> ---> K⁺ enters the hair cells from the scala media -----> depolarization and nerve firing result.
- POTASSIUM: It is unusual for this flow of K⁺ to result in depolarization. There is a large (+140mV) driving force for K⁺ to enter the cell, however.
- Glutamate: The IHC's probably use glutamate as an excitatory neurotransmitter to excite VIIIth afferents headed into the CNS.

HEARING LOSS

- Acoustic Neuroma: The most common type of CNS hearing loss, i.e. damage to the VIIIth nerve itself.
 - **Bell's Palsy** is common secondary symptom, as VIIIth nerve neuroma can also compress the VIIth nerve in the inner ear.
- Conduction Hearing Loss: Otosclerosis or Otitis Media.
- **WEBER'S TEST** can be used to distinguish conductive hearing loss from sensory hearing loss. Apply a vibrating tuning fork to the skull of a patient who has hearing loss in one ear.
- Sensorineural Hearing Loss: The most common type of hearing loss. Usually the problem is the hair cells, and the VIIIth nerve fibers are left intact.
 - CAUSES:
- Presbyacusia: Loss of hearing with age.

COCHLEAR IMPLANTS: Put in an implant that can provide direct stimulation to VIIIth-Nerve afferents. This can be done even if there is hair-cell deafness.

• This can restore some hearing but not usually speech perception. Best case scenario is speech perception is restored with practice.

MENIERE'S DISEASE:

- TRIAD OF SYMPTOMS: Patient will get ringing in ear \rightarrow "fullness" in ear, then hearing loss will drop off, then vertigo.
 - **Tinnitus** (ringing in ear)
 - \circ Fluctuating Hearing Loss
 - Episodic Vertigo
- ETIOLOGY: Idiopathic, Traumatic, Post-Syphilis, Viral
- PATHOPHYSIOLOGY:
- TRAUMA: Damage to the Endolymphatic Sac; you can't absorb endolymph fluid -----> fluid overload.
- Vestibular Membrane ruptures from buildup of inner fluid and pressure.
- **Hair Cell Toxicity** then occurs from mixing of endolymph and perilymph fluids. Some cell death. They think the membrane can heal itself, hence resultant hearing loss is only temporary.
- TREATMENT: Most common treatment is medicine designed to decrease the amount of inner ear fluid. • Salt balance / diuretics play a big role in treatment.
- Meclizine-Antivert, Valium, and Compazine can all be used as Vestibular suppressants.
- SURGICAL: Only if they don't respond well to medicine.
- Oscillopsia is a terrible visual side-effect where people bounce up and down.
- PROGNOSIS: *Progressive, untreated Meniere's disease leads to irreversible hearing loss.* Early treatment is therefore essential.

CENTRAL AUDITORY PATHWAYS

- Primary Sound Transmission: VIIIth Nerve -----> Cochlear Nucleus -----> Ventral and Dorsal Cochlear Nuclei -----> Inferior Colliculus where it SYNAPSES
- Secondary Sound Transmission: Inferior Colliculus -----> Brachium of the Inferior Colliculus -----> Medial Geniculate Body -----> Primary Auditory Cortex (Area 41).
- SOUND LOCALIZATION: The process of figuring out where in space a sound is originating from.
 SUPERIOR OLIVARY COMPLEX receives binaural (both ears) input, processes it, and sends a modifying signal to the Inferior Colliculus indicating the source of sound.
- **DUPLEX THEORY OF SOUND LOCALIZATION:** States that sound localization occurs by two different mechanisms, according to the frequency of the sound.
- Superior Colliculus (not Inferior): Plays a role in sound processing, in paying attention to peripheral space (same role as in visual processing). The auditory information and visual information are parallel to each other and occupy different layers in the same cortical columns within the Superior Colliculus.

AUDITORY EVOKED POTENTIALS: Place electrodes on head to measure the actual electrical activity generated by signals in the Auditory pathway.

- It is used as a substitute for normal hearing tests when a hearing test isn't possible: examples = infants, invalids, **malingerers** who behave like they are deaf when in fact they can hear.
- Can be used to diagnose some demyelinating diseases, such as Multiple Sclerosis, by studying the waveforms that are evoked in the auditory pathway.

TASTE AND SMELL

TASTE BUDS:

- Papillae:
 - **Circumvallate Papillae:** Located on the border between anterior 2/3 and posterior 1/3 of tongue. They contain the highest concentration of taste receptors.
 - Foliate Papillae: Located on the sides of the tongue and toward the back. Sparsely populated with taste buds.
 - Fungiform Papillae: Most abundant. Located on Anterior 2/3 of tongue.
- Taste Bud Structure: *Regeneration of Gustatory cells occurs*, so if you burn your tongue the sensation of taste will return!
 - \circ Receptor Cells have microvilli and are connected by tight junctions.
- Supporting Cells are immature receptor cells.
- Basal Cells are stem cells that produce new supporting cells, then new receptor cells.
- INNERVATION:
 - o Anterior 2/3: Facial Nerve (VII) -----> Geniculate Ganglion
- Posterior 1/3: Glossopharyngeal (IX) -----> Petrosal Ganglion
- Gustatory fibers on the Epiglottis are innervated by the Vagus (X) -----> Nodose Ganglion
- SOLITARY NUCLEUS in ipsilateral brain steam receives afferents from all of the above.
- DEFICITS:
 - Conductive Taste Loss occurs with lack of saliva. Saliva, dissolving the chemicals in food, is required for taste.
 - o Neural Taste Loss can occur, but it is rare because there are so many taste fibers.

ENSEMBLE ENCODING: Taste is perceived as a combination of the four basic tastes.

- Bitter: Back of the tongue
- Uses $IP_3 -----> Ca^{+2}$ as a second messenger.
- Sour: Posterior sides of the tongue.
- \circ Uses H⁺ to block K⁺ efflux -----> direct depolarization
- Sweet: Tip of the tongue
 - \circ Uses cAMP as a second messenger
- Salty: Anterior sides of the tongue
 - Opens Na⁺-Channels -----> direct depolarization

OLFACTION:

- Olfactory Cells: They are actually, themselves, neurons. They are original in that regard.
- **Basal Cells:** They differentiate directly into Olfactory Receptor cells, without the supporting cell intermediate.
- *Olfactory Neurons Regenerate*. Every time a new neuron is born, it must find its way through the **Cribriform Plate** and into the CNS.
- ENSEMBLE ENCODING: The system is even less organized than the taste system.
 - Specific: Each receptor cell binds to specific chemical stimuli.
 - Sensitive: Some receptors take an extraordinarily small amount of chemical stimuli to be activated.
- TRANSDUCTION: There is no synapse between the olfactory cells and the CNS. The olfactory receptors go straight to the CNS.
- Depolarization of the receptor occurs by a second messenger system: G-Protein -----> cAMP
- **OLFACTORY BULB:** Olfactory Receptors travel up through the Cribriform Plate to form the Olfactory Bulb.
- SYNAPSE occurs in Olfactory Bulb, in a GLOMERULUS, which are little balls of neuropil.
- **OLFACTORY TRACTS:** The axons of the second-order Mitral and Tufted Cells.
 - $\circ\,$ They then branch to form the Medial and Lateral Olfactory Stria.
- CNS OLFACTORY PROJECTIONS: The Olfactory Stria generally go through the Limbic System or Paleocortical System—not through the Thalamus.
 - Anterior Olfactory Nucleus: Part of the Olfactory Tract.
 - Septal Nuclei: They send signals -----> Hypothalamus.
- Piriform Cortex: Also called Paleocortex. They send signals -----> Entorhinal Cortex and Amygdala.
- Olfactory Tubercle: Base of the Olfactory Tract, part of Paleocortex. They send signal -----> Medial Dorsal Nucleus of Thalamus. This is conscious perception of odors.

ORBITOFRONTAL CORTEX: It receives the signals from the Thalamus and is responsible for the conscious perception of odor.